Has Health Capital Formation Cured 'Baumol's Disease'? – Panel Granger Causality Evidence for OECD Countries

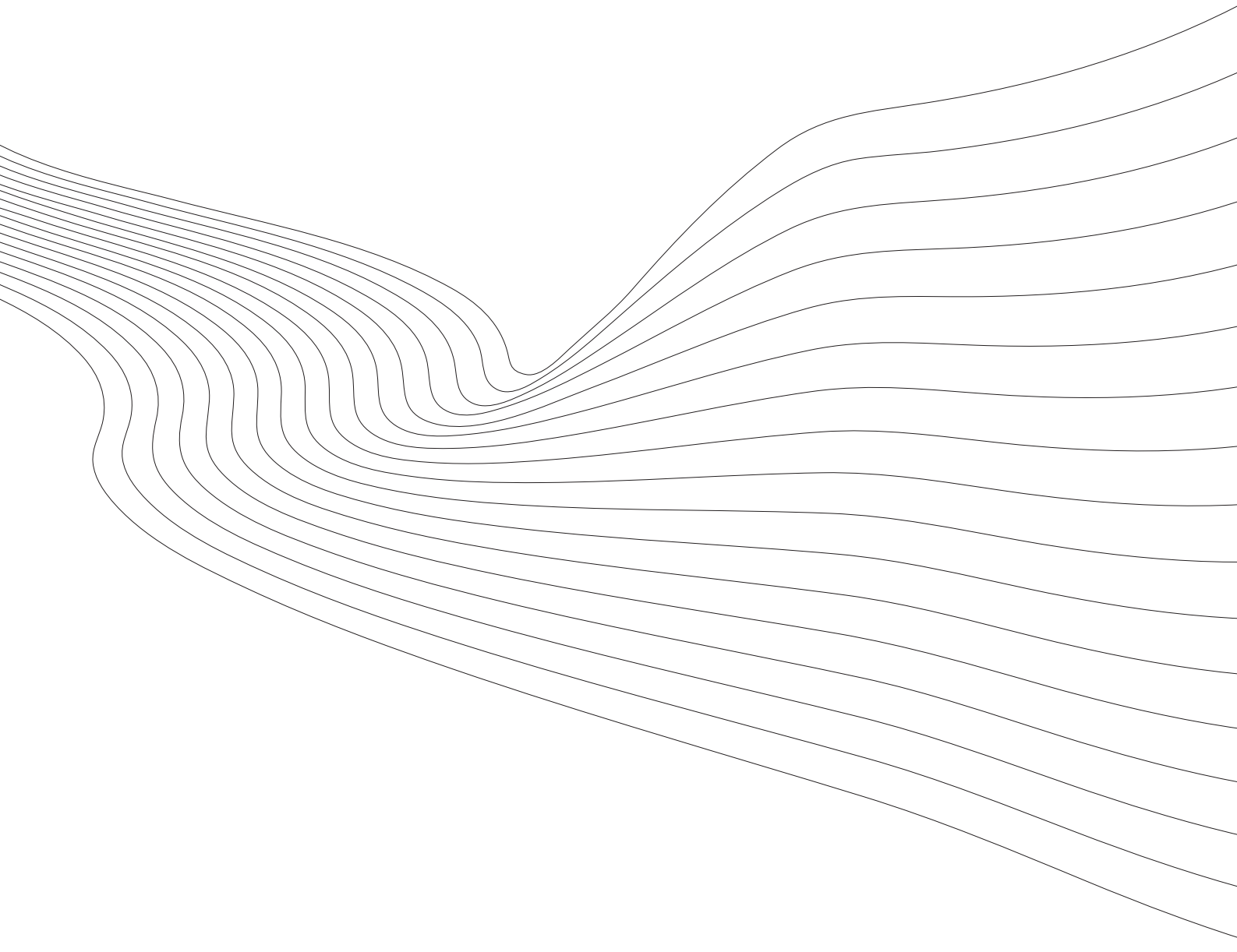
Panel Granger Causality Evidence for OECD Countries

KOF

KOF Working Papers

Has Health Capital Formation Cured 'Baumol's Disease'? – Panel Granger Causality Evidence for OECD Countries

Jochen Hartwig



No. 206
September 2008

ETH
Eidgenössische Technische Hochschule Zürich
Swiss Federal Institute of Technology Zurich

KOF

ETH Zurich
KOF Swiss Economic Institute
WEH D 4
Weinbergstrasse 35
8092 Zurich
Switzerland

Phone +41 44 632 42 39
Fax +41 44 632 12 18
www.kof.ethz.ch
kof@kof.ethz.ch

Has health capital formation cured 'Baumol's Disease'? – Panel Granger causality evidence for OECD countries

Jochen Hartwig
Swiss Economic Institute at ETH Zurich (KOF ETH)
Weinbergstrasse 35, 8092 Zurich
Switzerland

Telephone: +41-44-632-7331
Fax: +41-44-632-1218
E-mail: hartwig@kof.ethz.ch

Keywords: Human capital, health expenditure, 'Baumol's (Cost) Disease', panel Granger causality tests
JEL classification: C12; C23; I10; O41

Abstract

A large body of both theoretical and empirical literature has affirmed a positive impact of human capital accumulation in the form of health on economic growth. Yet Baumol (1967) has presented a model in which imbalances in productivity growth between a 'progressive' (manufacturing) sector and a 'nonprogressive' sector of the economy (of which health care forms an integral part) lead to perpetual expenditure shifts into the latter and, as a consequence, to a *decline* in overall GDP growth. Which of the two views has an empirical grounding is here tested by means of Granger causality analysis of a panel of 21 OECD countries. The results do not lend support to the hypothesis that health capital formation fosters economic growth in rich countries. They are more in line with the predictions of Baumol's model of unbalanced growth.

1. Introduction

Ever since Solow's (1957) growth model has been 'augmented' by human capital, the growth-enhancing role of human capital accumulation has been recognized. The pioneering empirical studies by Barro (1991) and Mankiw, Romer and Weil (1992) focused on the educational dimension of human capital, yet it has been known for long that human capital can also be accumulated by improving the health status of the population (Schultz, 1961, Mushkin, 1962). Weil's (2007) paper is the most recent in a chain of studies that have affirmed the growth-enhancing role of health capital formation empirically.

On the other hand, Weil (2007: 1295) also suggests that health's positive effect on GDP is strongest among poor countries. For rich countries, the existing empirical evidence on whether health capital formation stimulates GDP growth at all is mixed. For instance, while Rivera and Currais (1999a, 1999b, 2003) find a positive effect of health expenditure growth on productivity growth for OECD countries, Knowles and Owen (1995, 1997) as well as McDonald and Roberts (2002) reject the hypothesis that life expectancy is a statistically significant explanatory variable for productivity growth in high-income countries. Bhagrava et al. (2001) even estimate a negative effect of the adult survival rate on economic growth for the US, France, and Switzerland.[1] Acemoglu and Johnson (2006) reach a similar conclusion.

A possible reason why the impact of health capital formation on GDP growth might turn negative in the process of economic development is offered by Baumol's (1967) model of 'unbalanced growth'. According to this model, imbalances in productivity growth between a 'progressive' (manufacturing) sector and a 'nonprogressive' (services) sector lead to perpetual expenditure shifts into the latter. As a consequence of these expenditure shifts, the growth rate of overall GDP will decline. Interestingly, Baumol singles out the health sector as a cardinal example for an industry beset by which has to become known as the 'Cost disease'.

So far, the 'health and growth'-literature – focusing on the compelling supply-side arguments in support of a growth-enhancing role of health capital formation – has largely ignored the adverse structural change effect that Baumol (1967) calls attention to. On the other hand, the literature on structural change – for which Baumol's model of unbalanced growth is one of its cornerstones – has recently discovered health capital formation as a

[1] Drawing on current life tables, the adult survival rate measures the probability that a 15-year-old will reach the age of 60.

possible 'cure' for 'Baumol's (Cost) Disease'.[2] My aim in this paper is to bring these two strands of literature together and to investigate the causal relationship between health capital formation and GDP growth empirically. The empirical part of the paper focuses on rich countries because, for one thing, the growth-stimulating effect of health capital formation in poor countries is uncontested and, for another thing, 'Baumol's Disease' is a phenomenon that affects mainly developed economies. The econometric methodology I will use – the (dynamic) panel Granger causality framework – is relatively new and has (to the best of my knowledge) not been applied to study the relationship between health capital formation and economic growth before.

The paper is organized as follows. The next section introduces Baumol's model of unbalanced growth and discusses some theoretical and empirical controversies around it. Section 3 goes on to survey the state of the (empirical) 'health and growth'-literature. Section 4 opens up the empirical part of the paper introducing the data and methods to be used in the estimations, especially the methodology of (dynamic) panel Granger causality tests. Section 5 presents the results of the causality analysis including results of some robustness checks, and section 6 concludes.

2. Baumol's model of 'unbalanced growth' revisited

William J. Baumol's (1967) article '*Macroeconomics of unbalanced growth*' is widely regarded as a major contribution to the literature on structural change. Whereas the first champions of the 'tertiarization hypothesis', Allan Fisher (1935) and Colin Clark (1940), traced back the gradual shift in production from the agricultural to the manufacturing sector and onwards to the services sector mainly to changes in demand, Baumol offers a supply-side explanation.[3] In a nutshell, his model tells the following story. Productivity growth is higher in the 'progressive' (secondary) than in the 'nonprogressive' (tertiary) sector, but wages grow more or less the same in both sectors. Therefore, unit costs and also prices rise much faster in the tertiary sector than in the secondary. Demand for certain services, like health care for instance, is hardly price-elastic, hence consumers are willing to pay the higher prices. Therefore, an ever higher share of total expenditures will be channeled into the services sector – hence the 'Cost disease'. Also, since aggregate productivity growth is a weighted average of

[2] In a purely theoretical paper, Pugno (2006) sets up an endogenous growth model and hypothesizes that the growth-stimulating effect of health capital formation is likely to overcompensate the adverse sectoral shift effect when it comes to GDP growth.

[3] For a survey of the different 'tertiarization' theories, cf. Schettkat and Yocarini (2006).

the sectoral productivity growth rates with the weights provided by the nominal value added shares, the aggregate productivity growth rate will decline over time as the sector with the low productivity growth receives an ever-increasing weight. Thus, Baumol's model of unbalanced growth predicts a tendency for per-capita output in mature economies to stagnate.

The model is based on one fundamental assumption, namely that 'regular' growth in labor productivity can only occur in the 'progressive' sector. It is important to understand what the term 'regular' here means. For Baumol, *regular* productivity growth is the result of technological innovation which manifests itself in new capital goods. Capital goods are also responsible for economies of scale, being another source of productivity growth. *Regular* productivity growth is thus defined to depend on certain technological requirements. In the service industries, Baumol argues, and also in agriculture and construction, physical capital cannot be employed on the same scale as in manufacturing. These industries he therefore relegates to the 'nonprogressive' sector.[4] Baumol does not claim that increases in labor productivity are impossible in the 'nonprogressive' sector, only that this sector comprises "activities which, by their very nature, permit only sporadic increases in productivity" (Baumol, 1967: 416). In his model, Baumol abstracts from productivity increases in the 'nonprogressive' sector for simplicity.

Another simplifying assumption for the model is that labor is the only factor of production. Baumol (1967: 417) admits that this assumption is "patently unrealistic", yet it can be seen as a consequence of his basic assumption mentioned before because in the simplest version of the model, the 'nonprogressive' sector uses no capital. The employment of capital in the 'progressive' sector Baumol captures – at least over time – by postulating that labor productivity in this sector grows at an exogenous rate.

Next, Baumol assumes that nominal wages in both sectors are related in the long run. He simplifies further and assumes that they are equal. His final assumption is that nominal wages (in both sectors) rise to the same extent as labor productivity in the 'progressive' sector. This implies that the price level in the 'progressive' sector stays constant, whereas it rises in the 'nonprogressive' sector in order to keep the level of real wages in line with the productivity

[4] In the face of the all-round use of medical technology, the relegation of health care to the 'nonprogressive' sector may seem odd. Baumol points to the fact, however, that health care differs from manufacturing in that capital is here normally not used as a substitute for labor. "The bulk of our municipal expenditures", he writes, "is devoted to education which … offers very limited scope for cumulative increases in productivity. The same is true of police, of hospitals, of social services, and of a variety of inspection services. Despite the use of the computer in medicine …, there is no substitute for the personal attention of a physician …" (Baumol, 1967: 423). The issue of productivity growth in the health sector is also discussed in Hartwig (2008b).

level. The workers, regardless in which sector they work, buy goods and services from both sectors so that their respective real wages converge.

Equations (1) and (2) formally describe the production functions of the two sectors. Labor productivity in the 'nonprogressive' sector (1) stays constant, whereas it grows in the 'progressive' sector (2) at the constant rate *r*. Thus, output in the two sectors (Y_1 and Y_2) at time *t* is given by:

$$Y_{1t} = aL_{1t} \tag{1}$$

$$Y_{2t} = bL_{2t}e^{rt} \tag{2}$$

with L_1 and L_2 as quantities of labor employed in the two sectors and *a* and *b* as constants.

According to one of the assumptions just mentioned, the nominal wage (in both sectors) is given by:

$$W_t = We^{rt} \tag{3}$$

with *W* as an arbitrary starting value.

Equation (3) completes the model of 'unbalanced growth' already. This simple model has a couple of interesting implications which Baumol draws out. First, the 'Cost disease': equations (4) and (5) show that costs per unit of output in the 'nonprogressive' sector (C_1) tend towards infinity while they stay constant in the 'progressive' sector (C_2).

$$C_1 = W_t L_{1t} / Y_{1t} = We^{rt} L_{1t} / aL_{1t} = We^{rt} / a \tag{4}$$

$$C_2 = W_t L_{2t} / Y_{2t} = We^{rt} L_{2t} / bL_{2t}e^{rt} = W / b \tag{5}$$

Relative costs also tend towards infinity ($C_1 / C_2 = be^{rt} / a$). Under 'normal' circumstances – that is when prices rise in proportion to costs and when demand is price-elastic – the 'nonprogressive' sector will vanish. Baumol (1967: 421) invokes craftsmanship, fine restaurants, and theatres as examples of establishments that have either disappeared or retreated to luxury niches as a consequence of customers' unwillingness to tolerate the price increases that would have been necessary to cover rising costs.

Yet parts of the 'nonprogressive' sector produce necessities for which the price elasticity is very low. Baumol calls attention to education and health care as prime examples. To show what happens in these industries as a consequence of 'unbalanced growth', Baumol assumes that the relation of real output of the two sectors remains unchanged as in (6):

$$(b/a)\ \ Y_1 / Y_2 = L_1 / L_2 e^{rt} = K\,, \tag{6}$$

with $K = const$. If L (= L_1 + L_2) is the labor force, it follows:

$$L_1 = (L - L_1)Ke^{rt} \qquad \Leftrightarrow \qquad L_1 = LKe^{rt} / (1 + Ke^{rt}) \tag{7}$$

and $L_2 = L - L_1 = L/(1+Ke^{rt})$ (8)

From (7) and (8) we learn that, over the years ($t \to \infty$), L_1 tends towards L, and L_2 tends towards zero.

Finally, it can be shown what happens to the GDP growth rate under these conditions. Let I be an index for real GDP which is calculated as a weighted average of the value added of the two sectors:

$$I = B_1Y_1 + B_2Y_2 = B_1aL_1 + B_2bL_2e^{rt} \quad (9).$$

Then, if we insert (7) and (8) into (9) we get:

$$I = L(KB_1a + B_2b)e^{rt}/(1+Ke^{rt}) = Re^{rt}/(1+Ke^{rt}) \quad (10),$$

with $R = L(KB_1a + B_2b)$ (11).

Applying the quotient rule leads to:

$$\begin{aligned} dI/dt &= R[re^{rt}(1+Ke^{rt}) - Kre^{2rt}]/(1+Ke^{rt})^2 \\ &= rRe^{rt}/(1+Ke^{rt})^2 \end{aligned} \quad (12).$$

We can calculate the growth rate of real GDP as:

$$(dI/dt)/I = r/(1+Ke^{rt}) \quad (13).$$

It follows that over time ($t \to \infty$) the GDP growth rate drops asymptotically to zero *ceteris paribus*.[5]

Since its inception, the model of unbalanced growth has received much attention, both from economists working in the theoretical and empirical departments. In a theoretical vein, early critics focused on the almost complete neglect of the demand side in the model,[6] yet, since this is a common feature of growth models in the neoclassical tradition, this line of argument was not particularly damaging to Baumol's model. The supply-side core of the model didn't come under severe attack until Oulton (2001) was able to show that if the tertiary sector produces intermediate services instead of services to the final consumer, the aggregate productivity (and hence the GDP) growth rate may rise over time rather than fall. However, Sasaki (2007) vindicated Baumol's result of a tendency for the economy to stagnate, showing that the GDP growth rate will decline in the long run as long as *some* services are produced for final demand. This result is only subject to the condition that productivity growth in the 'nonprogressive' sector is indeed smaller than in the 'progressive' sector, as Baumol assumes.

[5] *Ceteris paribus* here especially means that L remains constant. If L grows at the rate m, then m must be added at the right hand side of (13).

[6] Cf. Harvey (1998) for a survey of the theoretical debate over Baumol's model during the first three decades after its inception.

Whether this assumption is valid has been investigated in a large body of empirical studies. Until recently, the empirical evidence was all but unanimously favorable to Baumol's model. Rowthorn and Wells (1987), Rowthorn and Ramaswamy (1997), Scarpetta et al. (2000), Wölfl (2003, 2005), and ECB (2006) are just a few examples for comparative multi-country studies that found productivity growth in manufacturing to be higher than in the aggregate of service industries. Fixler and Siegel (1999) confirm this finding in U.S. data. However, on the basis of a new dataset for the U.S., Triplett and Bosworth (2003, 2004, 2006) have raised doubts whether this 'stylized fact' is valid any longer. Taking output data from the Bureau of Economic Analysis' (BEA) industry output and input program and measuring labor input with BEA's series on persons engaged in production (full-time equivalents), Triplett and Bosworth (2004, table A–3) compute average annual productivity growth rates of 1.8 percent for the 'goods-producing industries' and of 2.3 percent for the 'service-producing industries' over the period 1995–2001. Consequently, they claim that 'Baumol's Disease has been cured' (Triplett and Bosworth, 2003, 2006). Still, since neither their 'goods-producing industries' match Baumol's 'progressive sector' nor their 'service-producing industries' match Baumol's 'nonprogressive sector', Triplett and Bosworth may have been too rash to dismiss 'Baumol's Disease', as Hartwig (2006, 2008a) argues. In fact, Bosworth and Triplett (2007) repeat their earlier calculations with *revised* U.S. National Accounts data and find their result of a higher productivity growth in the services sector than in the goods sector reversed.

As was mentioned in the introduction, Pugno (2006) has recently presented a challenge to Baumol's stagnationist outlook for the overall economy that does not rely on the possible non-validity of Baumol's fundamental assumption. Pugno argues that education and health care – the two services that Baumol (1967) uses as prime examples for 'nonprogressive' activities[7] – are on the other hand major contributors to the accumulation of human capital. Pugno hypothesizes that the growth-stimulating effect of health capital formation could even overcompensate the adverse sectoral shift effect when it comes to GDP growth. The next section shows that in the face of the existing empirical evidence, Pugno's optimism might seem warranted.

[7] Even Triplett and Bosworth who are convinced that 'Baumol's Disease' has been cured report very low productivity growth for health services (+0.9% per year over the period 1995-2001) and even a negative rate (–1.0%) for education (cf. Triplett and Bosworth, 2004, p. 350). There are, however, severe measurement difficulties with respect to productivity growth in these industries to which, for instance, Nordhaus (2008) calls attention.

3. Health capital formation and economic growth – a review of the literature

As was mentioned in the introduction, the notion of health as a component of human capital has been introduced in the early 60s already. In the famous Grossman (1972) model, the individual's desire to invest in his or her (depreciating) health capital stock explains the demand for health care. The growth literature has in turn identified investment in human capital as a driver of economic growth. So, not surprisingly, a number of studies have tried to determine the hypothesized contribution of health capital formation to growth empirically.

The first problem to solve in this literature is to measure health capital formation adequately. As can be seen from Table 1, which summarizes characteristics and results of a number of relevant studies in this field in chronological order, there are two main approaches.[8] Sixty percent of the studies rely on some measure of life expectancy or the adult survival rate in order to proxy health capital formation; another third choose health care expenditures (HCE) as a measure.[9] For the present study, I prefer the HCE approach for two reasons. First, health economists commonly trace back the lion's share of the increase in global life expectancy to factors such as economic development, better nutrition, and improvements in environmental conditions (where they have occurred) rather than to health care utilization (cf. Getzen, 1997: 330, Henderson, 1999: 142, Santerre and Neun, 2000: 69, Folland, Goodman and Stano, 2001: 118). Therefore, to proxy health capital formation with advances in life expectancy would loosen the link between health capital and health care that exists, for instance, in the Grossman model. Secondly, of course, by identifying health capital formation with health expenditure it is possible to build a bridge to Baumol's model of unbalanced growth, which predicts *expenditure* shifts towards the 'nonprogressive' sector.

< *Insert Table 1 around here* >

If we focus on the five studies in Table 1 that use measures of health expenditure as a proxy for health capital formation, we notice that all of them find a significantly positive impact of health capital formation on economic growth.[10] The same is true for the rest of the studies – with the exception of Webber (2002)[11] – when the full sample of countries is considered, although, as has been mentioned in the introduction, results (not shown in Table 1) are

[8] For similar compilations, cf. Tompa (2002) and Bloom, Canning and Sevilla (2004).

[9] Plus there is one study that uses calorie intake.

[10] The coefficients in Table 1 are not directly comparable since the models and samples differ.

[11] Weil (2007) doesn't use regression analysis to arrive at the estimate reported in Table 1.

sometimes different for the group of developed economies. Nevertheless, concerning the matter of dispute this paper sets out to resolve – whether health care expenditure growth through its impact on human capital formation fosters economic growth or, conversely, whether it leads to stagnation because it is the result of a 'Cost disease' that adversely affects the structure of the economy – the existing empirical evidence seems to support the former view.

However, if we take a closer look at the five studies mentioned, it appears that they do not yet provide enough evidence to close the case. All these studies use an augmented Solow growth modeling framework, which, in a cross-sectional design, does not allow for causal inferences. The positive contemporaneous association of GDP and HCE growth may be the result of 'reverse causation', as it is well known from the broad literature on the determinants of health care expenditure growth (recently summarized in Hartwig, 2008b) that GDP – or national income, respectively – is a very robust explanatory variable for health care expenditure.[12] Admittedly, Heshmati (2001) and Rivera and Currais in their various publications test for endogeneity using the Hausman (1978) test, yet no clear picture emerges. While Heshmati (2001) and Rivera and Currais (2004) cannot reject the hypothesis of exogeneity for health care expenditure, Rivera and Currais (1999a, 1999b, 2003) do find evidence for endogeneity and account for that by using instrumental variables in their estimations. It is unclear, however, whether the variables they propose as instruments are indeed appropriate since – as was just mentioned – the literature offers very little guidance as to the determinants of HCE growth (other than GDP growth). Also, Rivera and Currais' instrumental variables (IV) estimations are based on a cross-section of 24 OECD countries hence the number of observations is very limited.[13] At any rate, there still seems to be room for further research into the nexus between health care expenditure and economic growth. The present study aims at providing new evidence on the causal links between these two variables. In comparison to the earlier studies, more observations and a different methodology will be used. The next section will introduce the data as well as this different methodology, which is the (dynamic) panel Granger causality testing approach.

[12] Hoffmeyer and McCarthy (1994: 67) and Roberts (1999: 459) even suggest that GDP is *the only* robust determinant of health care expenditure that health economists have been able to uncover.

[13] Heshmati's (2001) data cover the period 1970-1992. He calculates four five-year averages and one three-year average, and he has 22 OECD countries in his panel. His database thus consists of 110 observations. Rivera and Currais (2004) also use a panel approach with five five-year averages over the period 1973-1993. For the 17 Spanish regions they look at, this yields 85 observations.

4. Methodology and data

Baumol's model of unbalanced growth predicts that structural change will cause the GDP growth rate to decline over time. Periods of strong increases in health care expenditure (HCE) are seen as episodes in which both employment and expenditures shift to a sector – or, insofar as HCE growth is symptomatic for an all-round 'Cost disease' – to sectors with low productivity growth. Later periods should then be characterized by lower GDP growth.

Otherwise, if Pugno (2006) was right, health expenditure forms human capital which, with a time-lag, should increase economic growth. In order to test which of these two hypotheses has an empirical grounding, we need a methodology that keeps track of the time-lags involved and that ascertains that causes, if we can identify them, precede effects. Both these aspects suggest choosing the methodology of Granger causality testing for the empirical part of this paper.

Granger's (1969) definition of causality has in the meantime become a standard analytical tool in applied econometrics. According to this definition, a stationary time series Y_t is said to 'cause' another stationary time series X_t if – under the assumption that all other information is irrelevant – the inclusion of past values of Y_t significantly reduces the predictive error variance of X_t. In econometric practice, whether Y_t Granger-causes X_t is typically tested by regressing X_t on its own lags and on lags of Y_t. If the lags of Y_t are found to be jointly statistically significant, then the hypothesis that Y_t Granger-causes X_t cannot be rejected. (Granger causality running from X_t to Y_t can be tested in the same way.)

More recently, the notion of Granger causality has found its way into panel econometrics. Most of the papers that have employed a panel Granger causality framework since around the beginning of the new millennium have investigated the causal nexus between certain variables and economic growth. For instance, there are papers that focus on 'investment and growth' (Attanasio, Picci and Scorcu, 2000, Podrecca and Carmeci, 2001) or, more narrowly, on 'foreign direct investment and growth' (Nair-Reichert and Weinhold, 2001, Laaksonen-Craig, 2004, Hsiao and Hsiao, 2006). The panel Granger causality approach has also been used to study the relationship between exports and growth (Chao and Buongiorno, 2002, Kónya, 2006), agriculture and growth (Tiffin and Irz, 2006), social security and growth (Lee and Chang, 2006) and financial development and growth (Hurlin and Venet, 2004, Miyakoshi and Tsukuda, 2004, Al-Awad and Harb, 2005). As far as I see, the present paper is the first to apply the panel Granger causality methodology to the field of 'health and growth'.

Since the methodology of panel Granger causality testing is not implemented identically in the literature just reviewed – for instance, not all papers impose the restriction that the

coefficients of the lagged X_t and Y_t variables are the same for all cross-section members – a few more words are necessary about how the method will be implemented here. First, as is quite standard in the literature just reviewed as well as in the broader literature on economic growth, the HCE and GDP level data will be transformed into five-year average annual growth rates (geometrical means) in order to eliminate the cyclical component. As the Granger causality tests require the data to be stationary, the resulting time series will be tested for the presence of unit roots, applying a battery of now standard panel unit root tests. When these tests fail to detect unit roots, the panel estimation models can be set up, for which the restriction of identical coefficients of the lagged X_{it} and Y_{it} variables will be imposed. Thus, I will estimate a time-stationary VAR model adapted to a panel context (as in Holtz-Eakin, Newey and Rosen, 1988) of the form:

$$X_{it} = \alpha_0 + \sum_{l=1}^{m} \alpha_l X_{it-l} + \sum_{l=1}^{m} \delta_l Y_{it-l} + \mu_i + u_{it} \qquad (14).$$

X_{it} and Y_{it} are the five-year averages of the growth rates of per-capita GDP and HCE, respectively. N countries (indexed by i) are observed over T periods (indexed by t). I allow for country-specific effects μ_i. The disturbances u_{it} are assumed to be independently distributed across countries with a zero mean. They may display heteroscedasticity across time and countries, though.

It is common in Granger causality studies to test whether causation runs in both directions. So although the main focus of this paper is on testing whether HCE growth Granger-causes GDP growth – and if so, with which sign – I will also estimate the equation:

$$Y_{it} = \beta_0 + \sum_{l=1}^{m} \beta_l X_{it-l} + \sum_{l=1}^{m} \gamma_l Y_{it-l} + \eta_i + v_{it} \qquad (15).$$[14]

It will be interesting to see whether the panel Granger causality methodology confirms health economists' 'stylized fact' that GDP is a significant explanatory variable for health care expenditure.[15]

[14] The assumptions are the same as for equation (14).

[15] As far as I see, the only two studies in this field of research that have used the Granger causality approach so far are the papers by Devlin and Hansen (2001) and Erdil and Yetkiner (2008). Devlin and Hansen find Granger causality running from GDP to HCE for 8 OECD countries out of 20 (only) in a sample of annual data that covers the period 1960–1987. Granger-causality running from HCE to GDP is also found for 8 countries. Erdil and Yetkiner's (annual) data cover a sample of 75 high to low income countries over the 90s. Their study finds bi-directional Granger-causality for 46 countries, Granger-causality running only from GDP to HCE for 12 countries and Granger-causality running only from HCE to GDP for 10 countries. Neither Devlin and Hansen nor Erdil and Yetkiner report the signs of their estimated coefficients.

Estimating equations (14) and (15) with pooled OLS presents an endogeneity problem since if the dummy variables (country-specific effects) affect GDP growth in (14) and HCE growth in (15) in one period, they presumably affected them in the previous period also (cf. Nickell, 1981). The first step into the direction of correcting this endogeneity problem in dynamic panels is to take the first difference of all variables and to thereby eliminate the individual effects. Still, there remains a correlation between the lagged dependent variable, which is now in differences, and the error term. Arellano and Bond (1991) have shown that the best way around this problem is to use lags of the dependent variable from at least two periods earlier (in levels) as well as lags of the right-hand side variables as instruments in a Generalized Method of Moments (GMM) estimator. Arellano and Bover (1995) and Blundell and Bond (1998) suggest to difference the instruments instead of the regressors in order to make them exogenous to the fixed effects. This leads from the 'difference' GMM to the 'system' GMM estimator (cf. Roodman, 2006). In the next section, I will present results using the OLS, Arellano-Bond one-step system GMM, and Arellano-Bond two-step system GMM estimators. For the latter, the finite sample correction recently proposed by Windmeijer (2005) will be applied.

Based on the estimation results, a conclusion on causality will be reached by running Wald tests on the coefficients of the lagged Y_t's to check whether they are jointly statistically different from zero.[16]

Data on health care expenditure are taken from the OECD Health database (in the version of October, 26, 2007). This database also contains demographic and economic entries so that all data needed for the purpose of the present paper can be gathered from this source.

Data on per-capita health care expenditure with a frequency of at least five years and a starting point around 1970 are available for 21 countries from the OECD Health database. These 21 countries are *Australia*, *Austria*, *Belgium*, *Canada*, *Denmark*, *Finland*, *France*, *Germany*, *Iceland*, *Ireland*, *Japan*, *Luxembourg*, *Netherlands*, *New Zealand*, *Norway*, *Portugal*, *Spain*, *Sweden*, *Switzerland*, the *United Kingdom*, and the *United States*.[17] All series end in 2005. From these data it is therefore possible to construct a balanced panel with 21 cross-sections and a time dimension of seven five-year-average growth rates. The panel thus consists of 147 observations (which is a clear improvement over the earlier empirical 'health and growth' literature that was discussed in section 3).

[16] Podrecca and Carmeci (2001) follow a similar approach to estimation and causality testing.

[17] The Danish series starts in 1971, the Dutch in 1972.

Data on real per-capita GDP (growth) are available for the same group of countries and period of time. In addition, I need data for the GDP deflator (which are also available from the OECD Health database) in order to deflate HCE – prior to calculating per-capita values and averaging growth rates, of course. Deflating nominal HCE with the GDP deflator is necessary to eliminate the upward trend in HCE that is due to purely monetary factors, or general inflation, respectively. If Baumol (1967) was right, then nominal HCE should grow more than nominal GDP – or HCE deflated by the GDP deflator should grow faster than real GDP – leading to a rise in the share of HCE in nominal GDP. This is precisely the 'Cost disease' point of view.[18] The next section will go on to present the results of the causality analysis, including results of some robustness checks.

5. Empirical results

A reasonable first step in empirical analysis is a visual inspection of the data. Figures 1 and 2 show the histograms of the per-capita GDP and per-capita HCE growth rates – both deflated by the GDP deflator – for my sample of 21 OECD countries. Obviously, health care expenditure has outgrown GDP. The median growth rate of real per-capita HCE is 3.6 percent, whereas the median growth rate of real per-capita GDP is only 2.2 percent.

< Insert Figures 1 and 2 around here >

Both per-capita GDP and HCE growth exhibit one large positive outlier. Ireland's per-capita GDP has grown by an amazing 8 ½ percent per year on average over the period 1995-2000. Even more extreme is Portugal's health expenditure growth between 1970 and 1975, a period in which real per-capita HCE more than doubled in this country. Outliers like these suggest that, as a robustness check, equations (14) and (15) should not only be estimated for the full sample of countries, but also by dropping each of the 21 countries in turn. Results of this robustness test will be presented below.

In order to test for Granger causality between GDP and health care expenditure, it is necessary that the two time series are stationary. Unfortunately, the available panel unit root tests are mainly designed for panels where both the time dimension and the cross section

[18] Health economists have indeed identified an 'excess health care price/cost inflation' (Huber, 1999), albeit without tracing it back to Baumol's model of unbalanced growth. This has only recently been done by Hartwig (2008b).

dimension are relatively large.[19] In panels such as mine with a time dimension of only 7 observations, the analysis can proceed only under restrictive assumptions like, for instance, dynamic homogeneity. This has to be kept in mind when interpreting the results of panel unit root tests reported in Table 2.[20] As the table shows, the tests reject the null hypothesis of non-stationarity for both variables. For what they are worth, these test results at least do not speak against proceeding to the Granger causality tests.

Since Granger causality test results are sensitive to the choice of lag length *m* in the time-stationary VAR model given by equation (14), it is important to specify the lag structure appropriately. I follow Miyakoshi and Tsukuda (2004) in estimating equation (14) with OLS and basing the choice of the optimal lag length on the Schwarz Information Criterion (SIC). Table 3 shows that – based on this criterion – the optimal lag length is two.

< Insert Table 3 around here >

Table 4 shows the results for estimating the VAR model (14) with OLS, with the Arellano-Bond one-step system GMM estimator and with the Arellano-Bond two-step system GMM estimator, respectively.[21] The OLS specification includes country-specific fixed effects (random effects were rejected by the Hausman test for correlated random effects.) The GMM specifications include period-specific effects. Lags of the dependent variable from at least two periods earlier as well as lags of the per-capita HCE growth variables serve as GMM-style instruments.[22] For the two-step estimator, the small sample correction proposed by Windmeijer (2005) has been implemented.

< Insert Table 4 around here >

The bottom of the table reports specification test results for the GMM estimations. The Sargan test is a test on whether the instruments are uncorrelated with the error term u_{it} (which they must be in order to be valid instruments). Table 4 shows that the null hypothesis is

[19] For a detailed account of panel unit root tests, cf. Breitung and Pesaran (2008).

[20] The table reports results for the panel unit root tests available in EViews. The estimations for this paper were done either with EViews (v. 6) or with Stata (v. 9).

[21] The Arellano-Bond one-step estimator uses the identity matrix as a weighting matrix. The two-step estimator weighs the instruments asymptotically efficient using one-step estimates.

[22] Roodman's 'xtabond2' command was used in Stata (v. 9) for the GMM estimations; and Roodman's (2006) examples geared my handling of the syntax.

accepted.[23] Note, however, that it is necessary to 'collapse' the set of instruments in order to achieve that the Sargan test accepts the over-identifying restrictions in the GMM estimations. While in the standard instrument matrix each instrumenting variable generates one column for each time period and lag available to that time period, Roodman (2006) proposes to 'collapse' the instrument set into a single column to limit the instrument count. This option is available in Stata (v. 9) and has been used here. The Arellano-Bond test accepts the hypothesis of no second-order autocorrelation in the disturbances of the first differenced equation.

The estimations consistently find *negative* coefficients for lagged per-capita health care expenditure. The second lag is always statistically significant. In the (OLS and) Arellano-Bond one-step estimation(s), the Wald test rejects the hypothesis that the coefficients of lagged per-capita HCE are jointly equal to zero. If they aren't, then growth in real per-capita health care expenditure Granger-causes real GDP growth with a negative impact.

There are at least two ways of interpreting this result. First, it could be seen as providing evidence against the model of endogenous growth that Pugno (2006) defends. In the standard neoclassical (exogenous) growth model, negative coefficients for the human capital variable are perfectly plausible within a Granger-causality framework. Starting from a steady state, a positive shock to health capital formation should boost GDP growth in the same time period. Later periods should then be characterized by lower growth as the growth rate falls back to its steady state level. Of course, while in the standard Solow model technological progress ensures that the steady state growth rate can remain positive, Baumol shows that when we assume 'unbalanced growth' in a two-sectoral model, the per-capita GDP growth rate will drop to zero despite technological progress (in the 'progressive' sector).

If we choose to stick to the endogenous growth model, however, then the finding of negative Granger-causality running from per-capita HCE to per-capita GDP growth undermines Pugno's (2006) conjecture that health capital formation might have cured the 'Cost disease' through its growth-stimulating effect (which overcompensates the adverse sectoral shift effect). This statement carries over to the Arellano-Bond two-step estimation which, although it does not find negative Granger-causality running from HCE to GDP growth at conventional significance levels, does not find any *positive* Granger-causality that would support Pugno's claim. Based on the evidence Table 4 reports, the research question

[23] The Sargan statistic, which is the minimized value of the one-step GMM criterion function, is not robust to heteroskedasticity or autocorrelation. The Hansen statistic (which is the minimized value of the two-step GMM criterion function) is robust.

that motivated this paper, namely whether health capital formation has cured 'Baumol's Disease', has to be answered in the negative.

Skeptics might object to the methodology employed to derive this result that it rests on the assumption that all information not included in the stationary VAR model is irrelevant. However, this assumption could be defended here on the grounds that the list of robust determinants of economic growth is not very long. Sturm and de Haan (2005), for instance, find only real capital formation and a couple of regional dummies (that are not relevant for my sample of OECD countries) to be significant and robust explanatory variables for GDP growth. To account for their findings, I added lagged per-capita growth in fixed investment to model (14) as a control variable. This didn't change the coefficients and Wald test results for HCE growth qualitatively.

Yet, there is still the possibility that the results Table 4 presents are driven by outliers. To check the robustness of these results, I therefore re-estimate equation (14) dropping each of the 21 countries in turn. Table 5 shows that the results are indeed to some extent sensitive to the exclusion of Portugal and Ireland. The Wald test nevertheless rejects the hypothesis that the coefficients of lagged per-capita HCE are jointly equal to zero at the 5 percent level.

< Insert Table 5 around here >

To conclude the empirical analysis, I test for Granger-causality running from GDP to HCE. As has been mentioned before, GDP – or national income, respectively – has been identified as the most important and robust explanatory variable for health care expenditure by health economists. Table 6 shows the results for estimating the VAR model (15).[24] As in the previous estimations, the GMM specifications include period-specific effects; and lags of the dependent variable from at least two periods earlier as well as lags of the per-capita GDP growth variables serve as GMM-style instruments. Again, the set of instruments was 'collapsed'. Still, the Sargan test does not accept the over-identifying restrictions at the 5 percent level. The Hansen test, however, does. The Arellano-Bond test clearly accepts the null of no second-order autocorrelation in the differenced residuals.

As can be seen from Table 6, the coefficients of lagged per-capita GDP are always positive and significant. The Wald test rejects the hypothesis that they are jointly equal to zero at conventional significance levels. Hence we can conclude that per-capita GDP growth Granger-causes per-capita HCE growth with a positive sign. This finding – which is robust to

[24] The Schwarz Information Criterion again suggests lag length two.

the exclusion of countries from the sample (cf. Table 7) – supports the existing literature on the determinants of health care expenditure growth.

< Insert Tables 6 and 7 around here >

6. Conclusion

The research question this paper poses – whether health capital formation has cured 'Baumol's (Cost) Disease' – can clearly be answered in the negative. No evidence was found that health capital formation through health care expenditure – that is the mechanism highlighted by the famous Grossman (1972) model – Granger-causes per-capita GDP growth with a positive sign in OECD countries. On the contrary, when per-capita GDP growth is regressed on its own lags and on the lags of per-capita health care expenditure in a panel Granger-causality testing framework, the coefficients for the lagged HCE growth are consistently negative. The statistical significance of the negative coefficients is not robust to the choice of the estimator (Arellano-Bond two-step vs. Arellano- Bond one-step system GMM), however.

When the other direction of Granger-causality – that is Granger-causality running from per-capita GDP growth to per-capita HCE growth – is tested, the results support health economists' long-standing 'stylized fact' that GDP determines HCE with a positive sign. My findings thus support the idea of some kind of self-stabilizing control cycle between GDP and health care expenditure. According to this idea, the strong HCE growth that occurred during the 50s and 60s in many OECD countries subsequently led to a decline in GDP growth because of the adverse structural change effect highlighted by Baumol's model. Declining GDP growth in turn attenuated HCE growth, and, as a consequence, GDP growth could rebound. This mechanism might help to understand why HCE growth in OECD countries declined temporarily in the 80s – a fact that Barros' (1998) model was unable to explain.

So how are my results related to the conventional wisdom that health is generally a good thing for economic growth?[25] First, it has to be remembered that my sample of countries consists of OECD countries only. Earlier studies (mentioned in the introduction to this paper) have occasionally also been unable to determine any positive influence of health on economic growth in rich countries. This does not mean that my results necessarily carry over to poor countries for which 'Baumol's Disease' will not become acute for a long time yet. Secondly,

[25] Weil (2007: 1266) quotes the World Health Organization's Commission on Macroeconomics and Health which sees 'powerful linkages' between health and long-term economic growth.

most studies that found a positive impact of health on economic growth used the aggregate production function approach and proxied health capital with some measure of the population's health status such as life expectancy at birth. Yet the health status can be improved in many ways; and indeed other determinants such as nutrition are generally regarded as more important for increasing life expectancy than health care utilization. My results point to a negative Granger-causality between health capital formation through health care expenditure and GDP growth. They do not preclude that increases in life expectancy through, say, better nutrition would stimulate economic growth.

Still, my results do present a challenge for the way the empirical growth literature generally proceeds, that is, by estimating an aggregate production function. If the output level is modeled to be a function of certain levels of input (such as health) then it is not surprising that increasing the input levels will raise output. Effects such as those highlighted by Baumol's model of unbalanced growth, namely that the process of raising certain input levels (such as health and education) can imply structural change that is unfavorable for overall economic growth might vanish in the estimates of total factor productivity growth.[26] For example, it might be the case that the health variable (life expectancy) has a positive sign in an estimated production function, but that a positive shock to health expenditure – even if it improves life expectancy – lowers total factor productivity to an extent that overcompensates the effect of the rise in health status on growth. Nevertheless, the positive sign of the health variable may mislead policymakers. To preclude this possibility, future research should find ways to cross-validate the conclusions of cross-country growth studies based on the aggregate production function approach.

References

Acemoglu, D., Johnson, S. (2006): Disease and development: the effect of life expectancy on economic growth, NBER Working Paper 12269.

Al-Awad, M., Harb, N. (2005): Financial development and economic growth in the Middle East, *Applied Financial Economics* 15(15), 1041–1051.

Arellano, M., Bond, S. R. (1991): Some tests of specification for panel data: Monte Carlo evidence and an application to employment equations, *Review of Economic Studies* 58(2), 277–297.

[26] Temple (1999: 150) also notes that "(t)he mention of structural change leads naturally to questioning the relevance of aggregate production functions".

Arellano, M., Bover, O. (1995): Another look at the instrumental variables estimation of error-components models, *Journal of Econometrics* 68(1), 29–51.

Attanasio, O. P., Picci, L., Scorcu, A. E. (2000): Saving, growth, and investment: a macroeconomic analysis using a panel of countries, *Review of Economics and Statistics* 82(2), 182–211.

Barro, R. J. (1991): Economic growth in a cross section of countries, *Quarterly Journal of Economics* 106(2), 407–443.

Barro, R. J., Sala-i-Marin, X. (1995): Empirical analysis of a cross section of countries, Ch. 12 in: *Economic Growth*, New York: McGraw-Hill.

Barros, P. P. (1998): The black box of health care expenditure growth determinants, *Health Economics* 7(6), 533–544.

Baumol, W. J. (1967): Macroeconomics of unbalanced growth: the anatomy of urban crisis, *American Economic Review* 57(3), 415–426.

Bhargava, A., Jamison, D. T., Lau, L. J., Murray, C. J. L. (2001): Modeling the effects of health on economic growth, *Journal of Health Economics* 20(3), 423–440.

Bloom, D. E., Canning, D., Sevilla, J. (2001): The effect of health on economic growth: theory and evidence, NBER Working Paper 8587.

Bloom, D. E., Canning, D., Sevilla, J. (2004): The effect of health on economic growth: a production function approach, *World Development* 32(1): 1–13.

Blundell, R., Bond, S. (1998): Initial conditions and moment restrictions in dynamic panel data models, *Journal of Econometrics* 87(1), 115–143.

Bosworth, B. P., Triplett, J. E. (2007): The early 21st century U.S. productivity expansion is still in services, *International Productivity Monitor* 14, Spring, 3–19.

Breitung, J., Pesaran, M. H. (2008): Unit roots and cointegration in panels, in: Matyas, L., Sevestre, P. (eds.): *The Econometrics of Panel Data*, 3rd ed., Berlin: Springer, pp. 279–322.

Chao, W. S., Buongiorno, J. (2002): Exports and growth: a causality analysis for the pulp and paper industries based on international panel data, *Applied Economics* 34(1), 1–13.

Clark, C. (1940): *The Conditions of Economic Progress*, London: Macmillan.

Devlin, N., Hansen, P. (2001): Health care spending and economic output: Granger causality, *Applied Economics Letters* 8(8), 561–564.

ECB (2006): Competition, productivity and prices in the Euro area services sector, European Central Bank Occasional Paper Series 44.

Erdil, E., Yetkiner, I. H. (2008): The Granger-causality between health care expenditure and output: a panel data approach, forthcoming in *Applied Economics* (DOI: 10.1080/00036840601019083).

Fisher, A. G. B. (1935): *The Clash of Progress and Security*, London: Macmillan.

Fixler, D. J., Siegel, D. (1999): Outsourcing and productivity growth in services, *Structural Change and Economic Dynamics* 10(2), 177–194.

Folland, S., Goodman, A., Stano, M. (2001): *The Economics of Health and Health Care*, 3rd ed., Upper Saddle River, NJ: Prentice Hall.

Getzen, T. (1997): *Health Economics: Fundamentals and Flow of Funds*, New York: John Wiley and Sons.

Granger, C. W. J. (1969): Investigating causal relations by econometric models and cross-spectral methods, *Econometrica* 37(3), 424–428.

Grossman, M. (1972): On the concept of health capital and the demand for health, *Journal of Political Economy* 86(2), 223–255.

Hausman, J. A. (1978): Specification tests in econometrics, *Econometrica* 46(6), 1251–1271.

Hartwig, J. (2006): Productivity growth in service industries – Has 'Baumol's Disease' really been cured?, KOF – Swiss Economic Institute Working Paper 155.

Hartwig, J. (2008a): Productivity growth in service industries – Are the transatlantic differences measurement-driven?, *Review of Income and Wealth* 54(3), 494–505.

Hartwig, J. (2008b): What drives health care expenditure? – Baumol's model of 'unbalanced growth' revisited, *Journal of Health Economics* 27(3), 603–623.

Harvey, M. C. (1998): Ecarts de productivité et »maladie des coûts«. Apports et limites du modèle de croissance déséquilibrée de William J. Baumol, *Revue économique* 48(2), 437–467.

Henderson, J. (1999): *Health Economics and Policy*, Cincinatti, OH: South-Western Publishing.

Heshmati, A. (2001): On the causality between GDP and health care expenditure in augmented Solow growth model, Stockholm School of Economics Working Paper in Economics and Finance 423.

Hoffmeyer, U. K., McCarthy, T. R. (eds.) (1994): *Financing Health Care*, Vol. 1, Dordrecht: Kluwer Academic Publishers.

Holtz-Eakin, D., Newey, W., Rosen, H. S. (1988): Estimating vector autoregressions with panel data, *Econometrica* 56(6), 1371–1395.

Hsiao, F. S. T., Hsiao, M.-C. W. (2006): FDI, exports, and GDP in East and Southeast Asia – Panel data versus time-series causality analyses, *Journal of Asian Economics* 17(6), 1082–1106.

Huber, M. (1999): Health expenditure trends in OECD countries, *Health Care Financing Review* 21(2), 99–117.

Hurlin, C., Venet, B. (2004): Financial development and growth: a re-examination using a panel Granger causality test, Laboratoire d'Economie d'Orléans Working Paper 18.

Jamison, D. T., Lau, L. J., Wang, J. (2005): Health's contribution to economic growth in an environment of partially endogenous technical progress, in: López-Casanovas, G., Rivera, B., Currais, L. (eds.): *Health and Economic Growth: Findings and Policy Implications,* Cambridge, MA: MIT Press, pp. 67–91.

Knowles, S., Owen, P. D. (1995): Health capital and cross-country variation in income per capita in the Mankiw–Romer–Weil model, *Economics Letters* 48(1), 99–106.

Knowles, S., Owen, P. D. (1997): Education and health in an effective-labour empirical growth model, *The Economic Record* 73(223), 314–328.

Kónya, L. (2006): Exports and growth: Granger causality analysis on OECD countries with a panel data approach, *Economic Modelling* 23(6), 978–992.

Laaksonen-Craig, S. (2004): Foreign direct investment in the forest sector: implications for sustainable forest management in developed and developing countries, *Forest Policy and Economics* 6(3/4), 359–370.

Lee, C.-C., Chang, C.-P. (2006): Social security expenditures and economic growth – A heterogeneous panel application, *Journal of Economic Studies* 33(5), 386–404.

Mankiw, N. G., Romer, D., Weil, D. N. (1992): A contribution to the empirics of economic growth, *Quarterly Journal of Economics* 107(2), 407–437.

McDonald, S., Roberts, J. (2002): Growth and multiple forms of human capital in an augmented Solow model: a panel data investigation, *Economics Letters* 74(2), 271–276.

Miyakoshi, T., Tsukuda, Y. (2004): The causes of the long stagnation in Japan, *Applied Financial Economics* 14(2), 113–120.

Mushkin, S. J. (1962): Health as an investment, *Journal of Political Economy* 70(5/2), 129–157.

Nair-Reichert, U., Weinhold, D. (2001): Causality tests for cross-country panels: a new look at FDI and economic growth in developing countries, *Oxford Bulletin of Economics and Statistics* 63(2), 153–171.

Nickell, S. (1981): Biases in dynamic models with fixed effects, *Econometrica* 49(6), 1417–1426.

Nordhaus, W. D. (2008): Baumol's diseases: a macroeconomic perspective, *The B.E. Journal of Macroeconomics* 8(1), Article 9.

Oulton, N. (2001): Must the growth rate decline? Baumol's unbalanced growth revisited, *Oxford Economic Papers* 53(4), 605–627.

Podrecca, E., Carmeci, G. (2001): Fixed investment and economic growth: new results on causality, *Applied Economics* 33(2), 177–182.

Pugno, M. (2006): The service paradox and endogenous economic growth, *Structural Change and Economic Dynamics* 17(1), 99–115.

Rivera, B., Currais, L. (1999a): Economic growth and health: direct impact or reverse causation?, *Applied Economics Letters* 6(11), 761–764.

Rivera, B., Currais, L. (1999a): Income variation and health expenditure: evidence for OECD countries, *Review of Development Economics* 3(3), 258–267.

Rivera, B., Currais, L. (2003): The effect of health investment on growth: a causality analysis, *International Advances in Economic Research* 9(4), 312–323.

Rivera, B., Currais, L. (2004): Public health capital and productivity in the Spanish regions: a dynamic panel data model, *World Development* 32(5), 871–885.

Roberts, J. (1999): Sensitivity of elasticity estimates for OECD health care spending: analysis of a dynamic heterogeneous data field, *Health Economics* 8(5), 459–472.

Roodman, D. (2006): How to do xtabond2: an introduction to "difference" and "system" GMM in Stata, Center for Global Development Working Paper 103.

Rowthorn, R., Wells, J. R. (1987): *De-industrialization and Foreign Trade*, Cambridge: Cambridge University Press.

Rowthorn, R., Ramaswamy, R. (1997): Deindustrialization: causes and implications, IMF Staff Studies for the World Economic Outlook, 61–77.

Santerre, R., Neun, S. (2000): *Health Economics: Theories, Insights, and Industry Studies*, Orlando, FL: Dryden Press.

Sasaki, H. (2007): The rise of service employment and its impact on aggregate productivity growth, *Structural Change and Economic Dynamics* 18(4), 438–459.

Scarpetta, S., Bassanini, A., Pilat, D., Schreyer, P. (2000): Economic growth in the OECD area: recent trends at the aggregate and sectoral level, OECD Economics Department Working Paper 248.

Schettkat, R., Yocarini, L. (2006): The shift to services employment: A review of the literature, *Structural Change and Economic Dynamics* 17(2), 127–147.

Schultz, T. W. (1961): Investment in human capital, *American Economic Review* 51(1), 1–17.

Solow, R. M. (1957): Technical change and the aggregate production function, *Review of Economics and Statistics* 39(3), 312–320.

Sturm, J. E., de Haan, J. (2005): Determinants of long-term growth: new results applying robust estimation and extreme bound analysis, *Empirical Economics* 30(3), 597–617.

Temple, J. (1999): The new growth evidence, *Journal of Economic Literature* 37(1), 112–156.

Tiffin, R., Irz, X. (2006): Is agriculture the engine of growth?, *Agricultural Economics* 35(1), 79–89.

Tompa, E. (2002): The impact of health on productivity: empirical evidence and policy implications, *The Review of Economic Performance and Social Progress* 2, 181–202.

Triplett, J. E., Bosworth, B. P. (2003): Productivity measurement issues in services industries: 'Baumol's Disease' has been cured, *Federal Reserve Bank of New York Economic Policy Review*, September, 23–33.

Triplett, J. E., Bosworth, B. P. (2004): *Productivity in the U.S. Services Sector – New Sources of Economic Growth*, Washington, D.C.: Brookings Institution Press.

Triplett, J. E., Bosworth, B. P. (2006): Baumol's disease has been cured: IT and multifactor productivity in U.S. service industries, in: Jansen, D. W. (ed.): *The New Economy and Beyond: Past, Present, and Future*, Cheltenham: Edward Elgar, pp. 34–71.

Webber, D. J. (2002): Policies to stimulate growth: should we invest in health or education?, *Applied Economics* 34(13), 1633–1643.

Weil, D. N. (2007): Accounting for the effect of health on economic growth, *Quarterly Journal of Economics* 122 (3), 1265–1306.

Windmeijer, F. (2005): A finite sample correction for the variance of linear two-step GMM estimators, *Journal of Econometrics* 126(1), 25–51.

Wölfl, A. (2003): Productivity growth in service industries: an assessment of recent patterns and the role of measurement, OECD – STI Working Paper 2003/7.

Wölfl, A. (2005): The service economy in OECD countries, OECD – STI Working Paper 2005/3.

Table 1: Macroeconomic growth studies with a focus on health

Study	Growth measure	Health measure	Countries and time period	Coefficient
Barro and Sala-i-Martin (1995)	Growth rate of per-capita GDP	Log of life expectancy at birth	87-97 countries 1965-85	0.064*
Knowles and Owen (1995)	Log difference of GDP per employed person between 1985 and 1960	Log of (80 years minus life expectancy at birth)	84 countries 1960-85	0.381*
Knowles and Owen (1997)	Log difference of GDP per employed person between 1985 and 1960	Log of (80 years minus life expectancy at birth)	77 countries 1960-85	0.582* – 0.797*
Rivera and Currais (1999a)	Log difference of GDP per employed person between 1990 und 1960	Log of the share of health expenditure in GDP	OECD-countries 1960-90	0.22* – 0.33*
Rivera and Currais (1999b)	Log difference of GDP per employed person between 1990 und 1960	Log of the share of health expenditure in GDP	OECD-countries 1960-90	0.21* – 0.22*
Bloom, Canning and Sevilla (2001)	Growth rate of per-capita GDP	Log of life expectancy at birth	104 countries 1960-90	0.04*
Bhargava et al. (2001)	Growth rate of per-capita GDP	Log of the adult survival rate	73-92 countries 1965-90	0.181* – 0.358*
Heshmati (2001)	Log difference of GDP per employed person between 1990 und 1970	Log of per-capita health expenditure	OECD-countries 1970-92	0.175*
Webber (2002)	Growth rate of GDP per employed person	Calorie intake per capita	46 countries 1960-1990	0.08 – 0.22
McDonald and Roberts (2002)	Log of GDP per employed person	Log of (80 years minus life expectancy at birth)	77 countries 1960-89	0.12*
Rivera and Currais (2003)	Log difference of GDP per employed person between 2000 und 1960	Log of the share of health expenditure in GDP	OECD-countries 1960-2000	0.18* – 0.26*
Rivera and Currais (2004)	Growth rate of GDP per employed person	Public health expenditure	17 Spanish regions 1973-93	0.13*
Bloom, Canning and Sevilla (2004)	Growth rate of per-capita GDP	Log of life expectancy at birth	104 countries 1960-90	0.04*
Jamison, Lau and Wang (2005)	Log of per- capita GDP	Log of the adult survival rate	53 countries 1965-90	0.50*
Weil (2007)	(Proportional reduction in variance of) log of GDP per capita	Adult survival rate (among others)	92 countries NA	0.099

* statistically significant at the 5% level

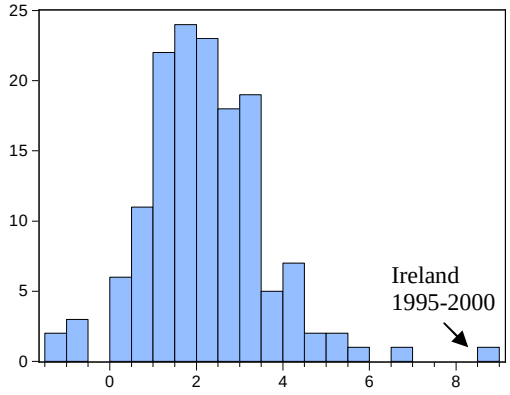


Figure 1: *Histogram of real per-capita GDP growth rates of 21 OECD countries (five-year averages, 1970-2005)*

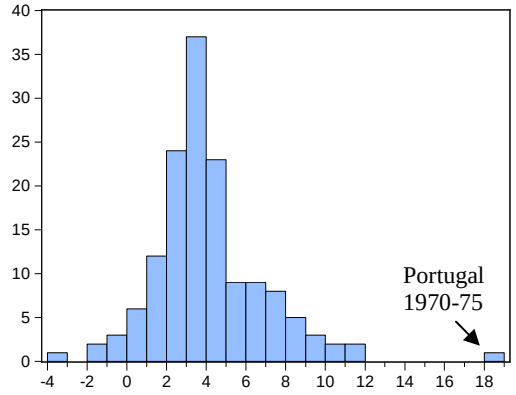


Figure 2: *Histogram of real per-capita HCE growth rates of 21 OECD countries (five-year averages, 1970-2005)*

Table 2: Panel unit root test results (21 OECD countries, 1970-2005)

	GDPRPC			HCERPC		
H_0: Unit root in level	Stat.	Prob.	Obs.	Stat.	Prob.	Obs.
Levin, Lin & Chu t*	–13.200	0.000	126	–17.128	0.000	126
Im, Pesaran & Shin W-stat	–5.007	0.000	126	–5.717	0.000	126
ADF – Fisher Chi-square	110.494	0.000	126	115.991	0.000	126
PP – Fisher Chi-square	129.068	0.000	126	168.481	0.000	126

GDPRPC = real per-capita GDP growth rates (five-year averages), HCERPC = real per-capita health care expenditure growth rates (five-year averages)

Note: Individual intercepts are included as exogenous variables in the test equations. For the first three tests listed in the table, maximum lags are automatically selected based on the Schwarz Information Criterion. The remaining test uses the Bartlett kernel for the Newey-West bandwidth selection. The probabilities for the Fisher tests are computed using an asymptotic Chi-square distribution. All other tests assume asymptotic normality.

Table 3: Optimal lag length for equation (14)

Lag	1	2	3
SIC	4.088	4.078	4.192

SIC = Schwarz Information Criterion

Table 4: Estimation results for equation (14)

	GDPRPC		
	(1)	(2)	(3)
	OLS	Arellano-Bond one-step GMM	Arellano-Bond two-step GMM
GDPRPC(−1)	–0.327***	0.346***	0.283**
	(0.113)	(0.125)	(0.111)
GDPRPC(−2)	0.241**	0.484***	0.477
	(0.116)	(0.139)	(0.317)
HCERPC(−1)	–0.074	–0.210**	–0.124
	(0.075)	(0.083)	(0.112)
HCERPC(−2)	–0.107**	–0.179***	–0.140*
	(0.047)	(0.057)	(0.068)
Number of obs.	105	105	105
Wald test (p-level)	0.061	0.002	0.149
Sargan test (p-level)	–	0.524	0.524
Hansen test (p-level)	–	–	0.458
AB test (p-level)	–	0.710	0.951

GDPRPC = real per-capita GDP growth rates (five-year averages), HCERPC = real per-capita health care expenditure growth rates (five-year averages)

Standard errors are in parenthesis. *, ** and *** denote significance at the 10, 5 and 1 percent level, respectively. Estimates for constant terms not shown. AB test = Arellano-Bond test for AR(2) in first differences.

Table 5: Robustness test – Cross-national stability of parameters of model (14): excluded countries

	GDPRPC										
	Australia	Austria	Belgium	Canada	Denmark	Finland	France	Germany	Iceland	Ireland	Japan
GDPRPC(–1)	0.350*** (0.122)	0.372*** (0.131)	0.347*** (0.128)	0.351*** (0.130)	0.325*** (0.121)	0.358*** (0.125)	0.345*** (0.128)	0.335*** (0.129)	0.316** (0.124)	0.070 (0.133)	0.397*** (0.124)
GDPRPC(-2)	0.472*** (0.139)	0.520*** (0.147)	0.487*** (0.142)	0.504*** (0.145)	0.434*** (0.136)	0.490*** (0.140)	0.487*** (0.142)	0.498*** (0.143)	0.509*** (0.144)	0.242* (0.127)	0.584*** (0.134)
HCERPC(-1)	–0.201** (0.083)	–0.249*** (0.094)	–0.208** (0.084)	–0.207** (0.088)	–0.179** (0.080)	–0.181** (0.087)	–0.215** (0.087)	–0.232*** (0.084)	–0.189** (0.084)	–0.165** (0.078)	–0.235*** (0.084)
HCERPC(-2)	–0.191*** (0.057)	–0.185*** (0.060)	–0.179*** (0.058)	–0.199*** (0.061)	–0.162*** (0.056)	–0.168*** (0.058)	–0.186*** (0.058)	–0.185*** (0.057)	–0.152*** (0.056)	–0.134** (0.052)	–0.211*** (0.056)
Number of obs.	100	100	100	100	100	100	100	100	100	100	100
Wald test (p-level)	0.001	0.002	0.003	0.002	0.005	0.007	0.002	0.001	0.008	0.017	0.000

	Luxembourg	Netherlands	New Zealand	Norway	Portugal	Spain	Sweden	Switzerland	U.K.	U.S.
GDPRPC(–1)	0.309** (0.132)	0.343*** (0.127)	0.355*** (0.124)	0.403*** (0.122)	0.355*** (0.129)	0.345*** (0.127)	0.355*** (0.129)	0.326*** (0.122)	0.348*** (0.128)	0.353*** (0.128)
GDPRPC(-2)	0.516*** (0.149)	0.505*** (0.141)	0.294** (0.137)	0.500*** (0.137)	0.439*** (0.147)	0.535*** (0.144)	0.497*** (0.143)	0.471*** (0.137)	0.487*** (0.142)	0.491*** (0.143)
HCERPC(-1)	–0.206** (0.083)	–0.237*** (0.084)	–0.211** (0.087)	–0.245*** (0.082)	–0.157* (0.080)	–0.216** (0.085)	–0.224*** (0.086)	–0.204** (0.083)	–0.207** (0.085)	–0.218** (0.087)
HCERPC(-2)	–0.195*** (0.055)	–0.204*** (0.058)	–0.188*** (0.057)	–0.166*** (0.056)	–0.136** (0.066)	–0.182*** (0.057)	–0.175*** (0.058)	–0.176*** (0.057)	–0.178*** (0.058)	–0.181*** (0.060)
Number of obs.	100	100	100	100	100	100	100	100	100	100
Wald test (p-level)	0.001	0.000	0.002	0.001	0.045	0.002	0.002	0.003	0.003	0.003

GDPRPC = real per-capita GDP growth rates (five-year averages), HCERPC = real per-capita health care expenditure growth rates (five-year averages)

The Arellano-Bond one-step GMM estimator was applied. Standard errors are in parenthesis. *, ** and *** denote significance at the 10, 5 and 1 percent level, respectively. Estimates for constant terms not shown.

Table 6: Estimation results for equation (15)

	HCERPC		
	(1)	(2)	(3)
	OLS	Arellano-Bond one-step GMM	Arellano-Bond two-step GMM
HCERPC(−1)	−0.244**	−0.189*	−0.139
	(0.099)	(0.102)	(0.106)
HCERPC(−2)	−0.382***	−0.336***	−0.326**
	(0.062)	(0.069)	(0.118)
GDPRPC(−1)	0.438***	0.493***	0.568***
	(0.149)	(0.153)	(0.163)
GDPRPC(−2)	0.332**	0.116	0.190
	(0.153)	(0.171)	(0.168)
Number of obs.	105	105	105
Wald test (p-level)	0.010	0.004	0.006
Sargan test (p-level)	–	0.035	0.035
Hansen test (p-level)	–	–	0.183
AB test (p-level)	–	0.801	0.781

GDPRPC = real per-capita GDP growth rates (five-year averages), HCERPC = real per-capita health care expenditure growth rates (five-year averages)

Standard errors are in parenthesis. *, ** and *** denote significance at the 10, 5 and 1 percent level, respectively. Estimates for constant terms not shown. AB test = Arellano-Bond test for AR(2) in first differences.

Table 7: Robustness test – Cross-national stability of parameters of model (15): excluded countries

	HCERPC										
	Australia	Austria	Belgium	Canada	Denmark	Finland	France	Germany	Iceland	Ireland	Japan
HCERPC(-1)	–0.177* (0.102)	–0.202* (0.108)	–0.201** (0.103)	–0.185* (0.108)	–0.188* (0.101)	–0.184* (0.106)	–0.190* (0.103)	–0.179* (0.104)	–0.175* (0.099)	–0.239** (0.108)	–0.189* (0.106)
HCERPC(-2)	–0.352*** (0.070)	–0.278*** (0.069)	–0.357*** (0.071)	–0.327*** (0.076)	–0.346*** (0.071)	–0.301*** (0.070)	–0.344*** (0.070)	–0.335*** (0.071)	–0.340*** (0.067)	–0.318*** (0.073)	–0.338*** (0.071)
GDPRPC(–1)	0.502*** (0.152)	0.532*** (0.151)	0.501*** (0.156)	0.492*** (0.160)	0.539*** (0.154)	0.528*** (0.152)	0.501*** (0.155)	0.465*** (0.160)	0.528*** (0.147)	0.478*** (0.185)	0.489*** (0.157)
GDPRPC(-2)	0.134 (0.172)	0.125 (0.168)	0.129 (0.173)	0.105 (0.178)	0.123 (0.172)	0.122 (0.170)	0.134 (0.172)	0.113 (0.178)	0.087 (0.170)	0.143 (0.176)	0.146 (0.169)
Number of obs.	100	100	100	100	100	100	100	100	100	100	100
Wald test (p-level)	0.003	0.001	0.004	0.005	0.002	0.002	0.004	0.011	0.001	0.026	0.007

	Luxembourg	Netherlands	New Zealand	Norway	Portugal	Spain	Sweden	Switzerland	U.K.	U.S.
HCERPC(-1)	–0.166* (0.101)	–0.199** (0.102)	–0.203* (0.112)	–0.191* (0.106)	–0.145 (0.101)	–0.183* (0.102)	–0.194* (0.103)	–0.198* (0.104)	–0.183* (0.103)	–0.221** (0.105)
HCERPC(-2)	–0.342*** (0.067)	–0.370*** (0.070)	–0.322*** (0.074)	–0.339*** (0.072)	–0.367*** (0.082)	–0.341*** (0.068)	–0.342*** (0.070)	–0.341*** (0.071)	–0.342*** (0.071)	–0.332*** (0.072)
GDPRPC(–1)	0.477*** (0.161)	0.485*** (0.153)	0.436*** (0.160)	0.516*** (0.158)	0.390** (0.160)	0.554*** (0.153)	0.499*** (0.155)	0.527*** (0.153)	0.489*** (0.155)	0.493*** (0.154)
GDPRPC(-2)	0.091 (0.182)	0.121 (0.171)	0.066 (0.177)	0.132 (0.178)	0.058 (0.183)	0.229 (0.173)	0.130 (0.173)	0.146 (0.171)	0.104 (0.173)	0.110 (0.172)
Number of obs.	100	100	100	100	100	100	100	100	100	100
Wald test (p-level)	0.006	0.005	0.020	0.004	0.041	0.001	0.004	0.002	0.005	0.004

GDPRPC = real per-capita GDP growth rates (five-year averages), HCERPC = real per-capita health care expenditure growth rates (five-year averages)

The Arellano-Bond one-step GMM estimator was applied. Standard errors are in parenthesis. *, ** and *** denote significance at the 10, 5 and 1 percent level, respectively. Estimates for constant terms not shown.